NATIONAL BUREAU OF STANDARDS REPORT

6317

Progress Report

on

MICROSTRUCTURE OF THE HUMAN TOOTH

by

W. H. Jennings
A. F. Forziati
F. L. Losee

U. S. DEPARTMENT OF COMMERCE
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The Primary Caries Lesion

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W. H. Jennings*
A. F. Forziati**
F. L. Losee***

* Physicist, Naval Medical Research Institute, Bethesda, Md.
** Research Associate, American Dental Association Research Division, Dental Research Section, National Bureau of Standards.
*** Dentist, Naval Medical Research Institute, Bethesda, Md.

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MICROSTRUCTURE OF THE HUMAN TOOTH

The Primary Caries Lesion*

Abstract

This report concerns the primary or pre-clinical enamel lesion which may be regarded as the earliest structural alteration of the enamel by the caries process. This lesion exhibits altered birefringence, enhanced fluorescence, and no loss of mineral. Based on clinical and laboratory observations, the following mechanism of the formation of this primary lesion is presented: Some organic agent initiating from the accumulation of oral material on the enamel surface diffuses into the enamel and produces the structural alteration described above. The advanced lesion is similar to the primary lesion, but may exhibit several geometrical forms which result from the influence of structural features on the microscopic progress of the lesion. The hypercalcified outer layer of human enamel caries is demonstrated by optical techniques and by two simple experiments.

1. INTRODUCTION

Although the mechanics of caries has been widely investigated, nearly all of the carious lesions studied have been detectable by some method of clinical examination. This report concerns a primary or pre-clinical enamel lesion which may be regarded as the earliest structural alteration of the enamel by the carious process.

Within the past century four basic concepts of the mechanics of enamel degradation have been considered. One early group of investigators placed the initial action of the carious process in the organic component of enamel. This viewpoint was well stated in 1881 by Underwood and Milles [1], who wrote, "Caries is absolutely dependent upon the presence and proliferation of organisms ..... these organisms attack first the organic material and feeding upon it create an acid which removes lime salts."

The inverse concept was set forth when Miller [12, 13] reported: "..... acids, especially those generated in the oral cavity by fermentation, are factors which by no means should be overlooked in a discussion of the cause of caries, since by softening the tissue of the tooth, they expose it to the action of other agents which follow after."

* This is the second in a series of papers on the microstructure of the human tooth (see ref. 26).

In 1948 the Michigan Workshop on Caries [18] characterized the carious process as a decalcification by acids formed by the action of microorganisms on carbohydrates. However, the decalcification was considered to be accompanied or followed by disintegration of the organic fraction.

Most recently the dual mechanism of proteolysis-chelation has been proposed. Schatz [19] in 1957 formulated the concept as "..... two interrelated reactions occurring simultaneously in enamel: (a) microbial destruction of the organic matrix which is largely keratin and (b) loss of apatite through dissolution by organic chelators some of which originate as matrix degradation products."

The use of only four examples to summarize the etiology of caries may appear presumptuous, but these illustrations do encompass most concepts to be found in the literature. In review these are:

1) Primary action on the organic fraction [1-11].

2) Primary action on the inorganic fraction [12-17].

3) Primary action on the inorganic fraction followed or accompanied by action on the organic fraction [18].

4) Proteolysis-chelation .... Nearly simultaneous degradation of both fractions [19].

From a clinical standpoint caries is not, in general, an allover effect; that is, it affects only limited areas of the tooth rather than the entire exposed surface. Furthermore, it is most frequently found in regions where the surface of the tooth is neither naturally nor artificially cleansed.

As investigators, we seek a mechanism for the initial carious lesion which is consistent with these clinical observations and the state of the tooth in the oral cavity. Known conditions in the oral cavity include:

1) Presence of microorganisms.

2) Oral inter- and extracellular enzyme systems.

3) Organic degradation products.

4) Accumulations of oral material on the surface of the tooth.

5) Diffusibility or permeability of intact enamel [20, 21, 22].

6) Alteration of the enamel only in presence of some agent [23].

Within this framework we propose the following hypothesis:

> Without entering into the definition or existence of the dental "plaque" we can state that oral debris, exudates, and microorganisms accumulate on the surface of the enamel. Such an accumulation may be highly localized or it may spread out over a considerable area of the smooth surface. The enamel under some of the amassed material undergoes a subtle change not demonstrable clinically but which is preparatory and essential to the destruction of the enamel by the carious process.

To prove this hypothesis the surface of the enamel must be carefully investigated in an attempt to detect the subtle change postulated. Since such an investigation involves the analysis of areas that are not dimensionally amenable to chemical techniques, a battery of optical techniques are utilized. This approach has been shown to be extremely effective by Darling [24], Gustafson [25], and Losee [26] and their collaborators in that the information obtained from a single technique may be extended by correlation with one or more other techniques applied to the same specimen.

2. OBSERVATIONS

Several hundred untreated teeth have been routinely serial sectioned in connection with our investigations of the microstructure of the human tooth. Most of these were third molars from males between the ages of 18 and 25 and many were classified "caries-free." When studying these mineralized sections under the polarizing microscope, a rather common observation is the small irregular area illustrated in Figure 1. This area shows a marked change in birefringence and appears to be contained within the incremental lines. Figure 2 represents a more advanced condition, but even here little or no surface change can be demonstrated by the clinician in ordinary light and nothing can be detected with an explorer.

In Figure 3 the initial lesion is again represented by an altered birefringence, and in addition surface accumulation of material can be seen. This area also possesses an enhanced fluorescence when irradiated with a band of ultra-violet centered at 3650 Å, (Figure 4); its radiodensity as shown by a microradiograph (Figure 5) indicates no detectable loss of mineral. Briefly then,

the initial action does not involve a demonstrable demineralization. Rather, it is characterized by altered birefringence and enhanced fluorescence.

A change in form birefringence due to alteration of the organic matrix of the enamel can occur in the absence of any demonstrable degradation of mineral constituents. This must be the case in the initial enamel lesion, for the probability of degraded mineral matter remaining at the enamel surface is very small. Thus the radiopacity of the microradiograph is characterizing intact mineral forms, and the altered birefringence indicates changes in the organic structures of the enamel.

The remaining characteristic of the initial lesion, its enhanced fluorescence, permits speculation. First, let us consider a system in which the microorganisms in the accumulated surface material are releasing an enzyme which is capable of infiltrating the enamel. As enzymes are proteins, it is possible that they may fluoresce when exposed to near ultraviolet radiation. The presence of enzymes in the enamel would thus enhance the normal enamel fluorescence. Another possibility in the same system depends upon the action of the enzyme on the enamel. Such action might involve the creation of some highly fluorescent organic compound or the destruction or removal of a substance capable of quenching the fluorescence. Also a more efficient fluorescence system might be produced by the creation of a mineral-organic bond or a local pH shift in a favorable direction.

An observation made during the extraction of bone with ethylenediamine to remove organic material shows a correlation between intensity of fluorescence and nitrogen content of the bone.* During the first two hours of extraction, both quantities increase; thence, an exponential decay occurs in both. The increased nitrogen is explainable by the method of calculation which is based upon dry weight. The initial ethylenediamine extraction removes loosely combined components, thus reducing weight rapidly without reducing the collagen (keratin in enamel) fraction significantly. This means that the fluorescence increase is large compared to the change in total nitrogen, and hence total organic material. If ethylenediamine acts in the same manner as the agent involved in the initial enamel lesion, all of the above mechanisms of fluorescence enhancement remain possible. If, however, it is a general and not a specific action of the ethylenediamine that is responsible for the change, the number of possible mechanisms is reduced. Now if the ethylenediamine acts merely as a solvent, it could remove a quenching substance or it could reduce concentra-

* Note: These data will be presented elsewhere.

tion quenching. If ethylenediamine acts to form molecular complexes, it could inactivate a quenching substance or it could form a fluorescent complex.

The above considerations are based on a postulated enzyme. The same arguments can be set forth with the same validity if instead of an enzyme the agent postulated is the metabolic by-product (possibly a chelator or organic acid) of a microorganism, a degradation product of the surface accumulation, or a product of the degradation of the organic enamel matrix [27, 28, 29].

From the macroscopically undetectable initial lesion progress of the classical white spot is a matter of degree and of stratification of the lesion into distinct zones [25]. The birefringence now shows a number of distinct changes corresponding to particular zones (Figure 6). The broad band of fluorescence (Figure 7) is no longer uniform due to irregular areas of low intensity towards the surface. These areas appear radiolucent in the microradiograph (Figure 8); thus they have sustained a loss in mineral.

At this stage the clinician observes a shiny unbroken surface, covering an opaque white area, which is not penetrated by his explorer [30]. If he were to expose the tooth to ultraviolet radiation, the area would appear black against the natural fluorescence of the enamel. Thus with advance of the lesion a stage of demineralization is reached that is characterized by lack of mineral, loss of birefringence, and loss of fluorescence.

It is of interest to note that the "early" enamel lesion is observed in a variety of geometrical forms [31]. Figures 9 and 10 illustrate a shallow, localized lesion. Figures 11 and 12 illustrate the classic lesion -- the truncated cone, which is localized and penetrating. Figure 13 illustrates a generalized, shallow lesion on the buccal surface.

The different shapes of the macroscopic lesion derive from the progress of the lesion on the microscopic level. In some cases (Figure 14) microradiographs reveal a fairly uniform progression of the demineralization over a very large area of a crown. In a more localized lesion (Figure 15) there appears to be no preferred direction of attack, while another lesion (Figure 16) shows a remarkable demineralization along one striae of Retzius.

Each of these microradiographs demonstrates a mineralized surface layer above the lesion [7, 24, 25, 32, 33]. This cannot be discounted as a "Macke" line effect because the zone is not confined to a uniform region following the contour of the enamel surface. In a polarization photomicrograph (Figure 17) similar to Figure 6, a calcified zone is seen at the surface above the

lesion. Because the retardation of this zone is different from that of the adjacent unaffected enamel, it cannot be regarded as unaltered.

The existence of this surface layer can be demonstrated on the macroscopic lesion. The distal contact point lesion illustrated in Figure 18 was tapped with a chisel exposing the softer chalky white material seen in Figure 19. Still another demonstration of this outer layer can be made by placing a thin section into an ethylenediamine tetracetic acid decalcifying chamber and observing the progress of the mineral removal (Figure 20). When the midpart of the lesion is almost completely removed, a dense surface layer still remains. Also note that the front of the lesion shows a zone of relatively higher mineral content.

3. DISCUSSION

The hypercalcified outer layer of human enamel caries was described as early as 1934 by Thewlis [32]. In 1937 Pincus [7] described this intact layer above the lesion, and Applebaum [33] presented radiographic evidence for the layer similar to Darling's[24] and the material in Figures 16, 17 and 18. This aggregate of evidence together with many examples from polarization photomicrography similar to Figures 6 and 17 and the demineralization observations negate any consideration of this layer as an artifact. As to the nature of this layer, it has been shown above that it cannot be considered unaltered enamel. Gustafson [25] feels that the minerals leached out of enamel at the active carious front may diffuse either ahead of the front or in the opposite direction; and this surface layer derives from reprecipitated mineral salts.

As the clinical lesion evolves, the birefringence and fluorescence are entirely lost and so is the mineral component of the area. At the front of the lesion, a pattern similar to the initial lesion is observed, but it is complicated by the presence of a hypermineralized zone presumably due to diffusing mineral ions.

The evidence presented in this report supports a hypothesis for the mechanism of the initial enamel lesion. This mechanism has many of the characteristics of the primary enamel degradation in the clinically observable lesions and indeed may be identical. The mechanism has as its agent some organic material initiating from an accumulation of oral material on the enamel surface. This agent diffuses into the enamel and produces an effect demonstrable as change in birefringence, an enhanced fluorescence, and no loss in mineral. The nature and action of the agent is obviously important to the elucidation of the detailed carious process. The technique of tagging with radioisotopes and autoradiography present the possibility of obtaining this highly specific information.

4. SUMMARY

1. A new hypothesis of the formation of a primary carious lesion is presented. This states that the surface accumulation of organic material is the source of some organic agent which enters the enamel and produces a change not demonstrable clinically but which is preparatory to the destruction of the enamel.

2. The carious lesion is shown to exist in several geometrical forms quite different from the "classical" truncated cone, and the form of the lesion is influenced by structural features on the microscopic level.

3. The hypermineralization of the surface of enamel caries is substantiated.

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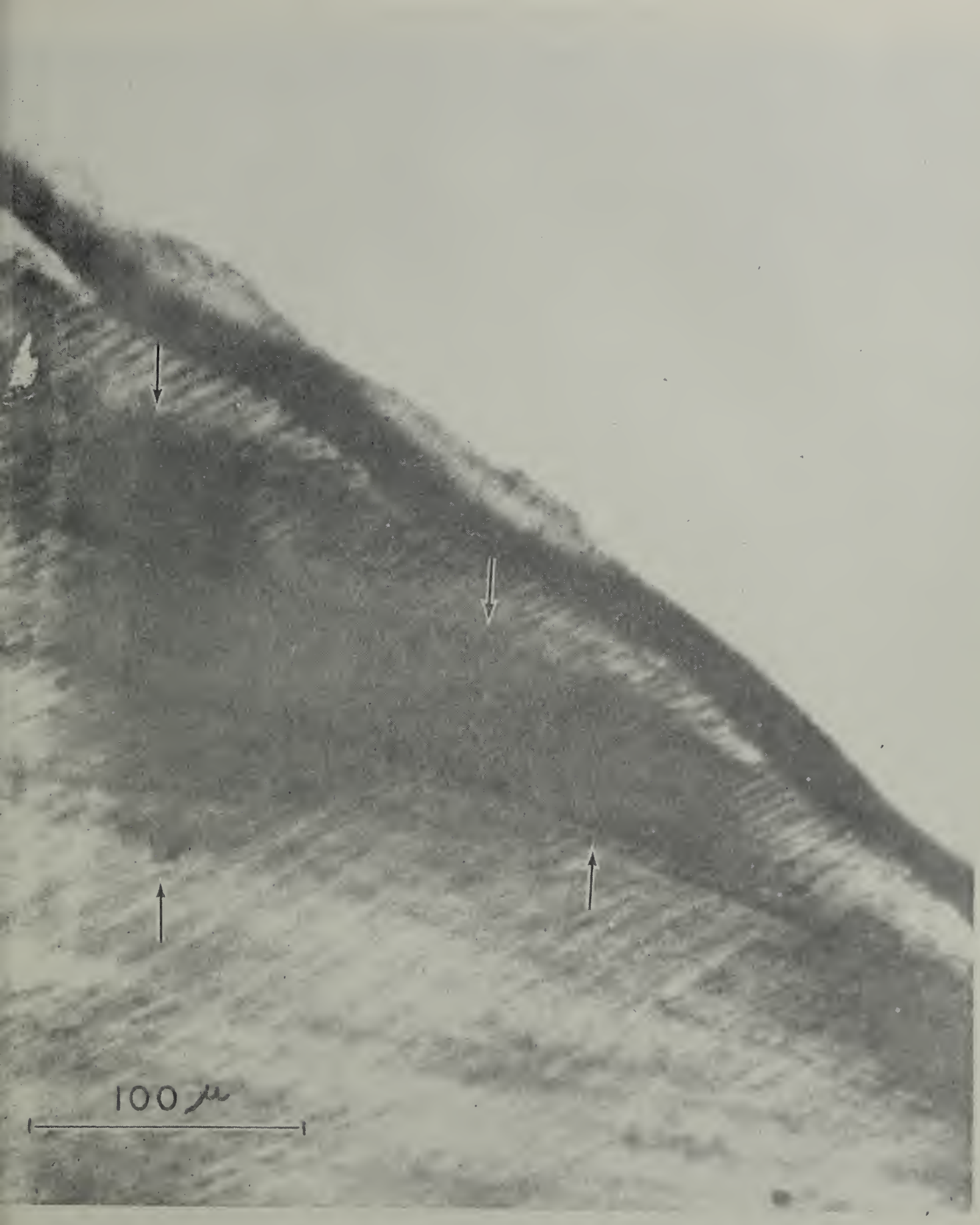

Figure 1. Polarized light, Nicols crossed, quartz wedge set for first order red. An irregular area of altered birefringence bounded by the incremental lines.

Figure 3. Polarized light, Nicols crossed, first order red. The initial lesion demonstrated by an altered birefringence. Note the surface accumulation of material above the lesion.

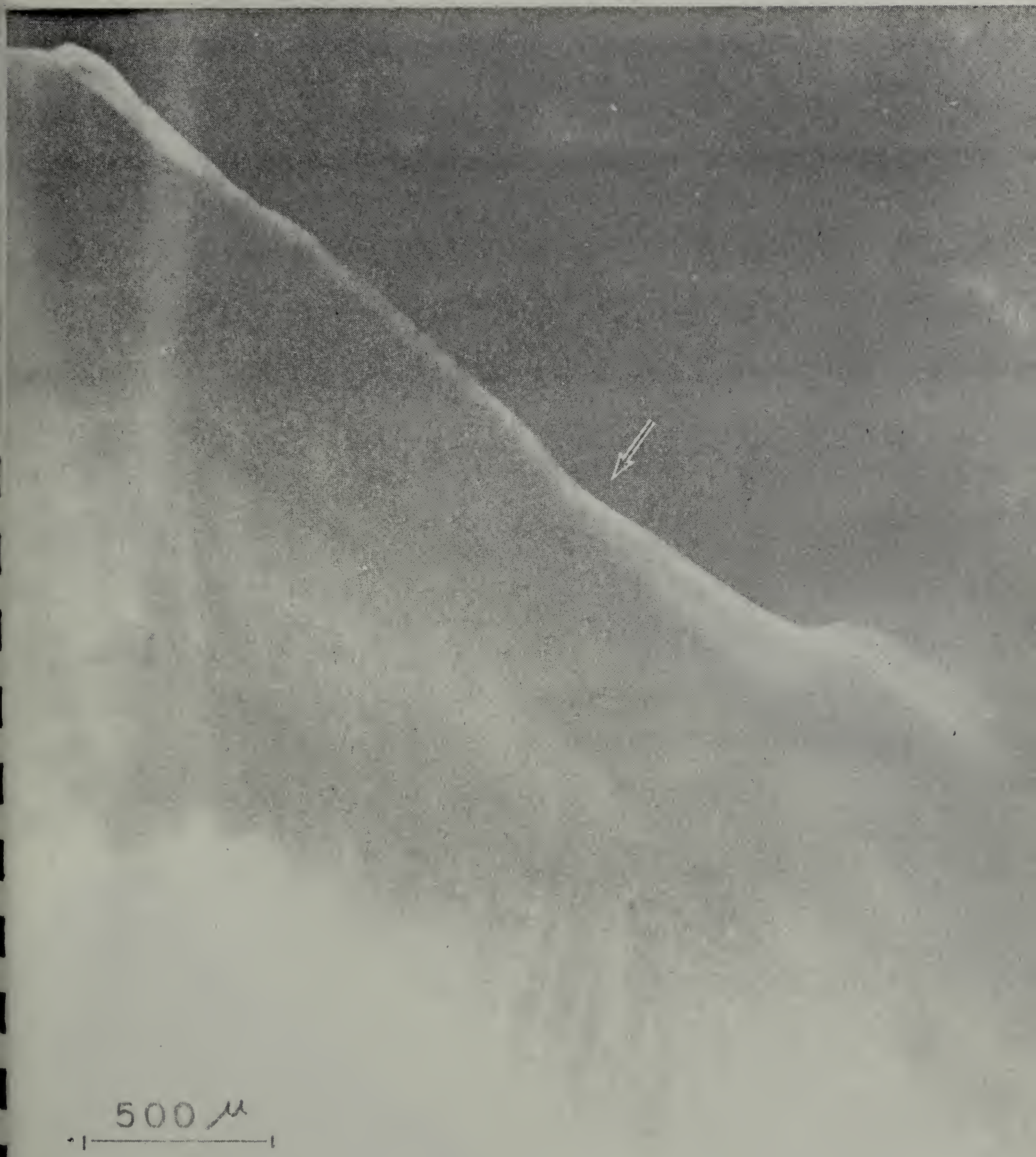

Figure [illegible]. Photomicrograph, [illegible] section. Same area as Figure [illegible], area of lesion shown at enhanced fluorescence.

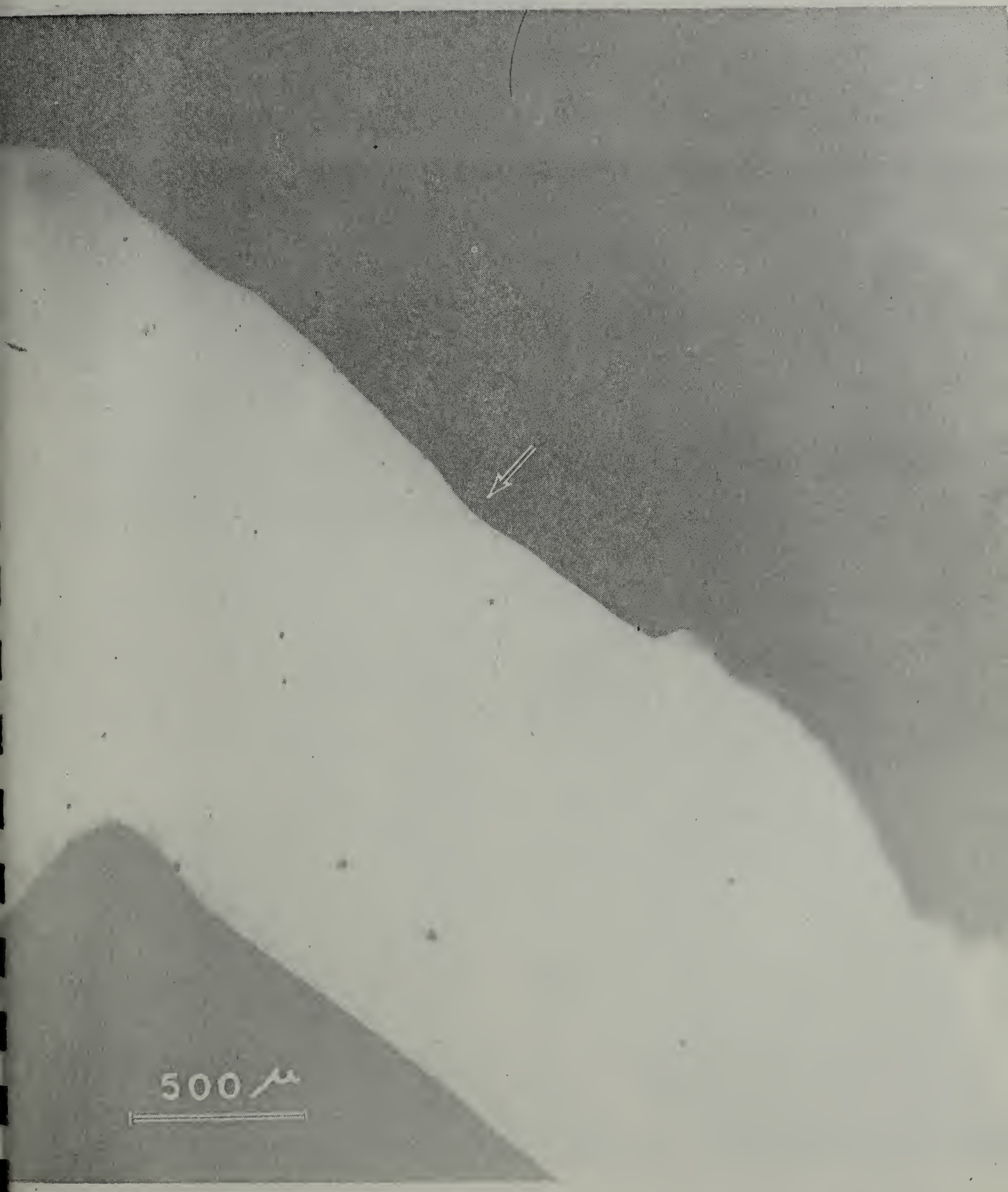

Figure 5. Microradiograph. Same area as Figures 3 and 4. Radiodensity of the lesion area indicates an demineralization.

Figure 6. Polarized light, Nicols crossed, first order red. The clinically observable stratified lesion.

Figure 7. Fluorescence micrograph. Same area as Figure 6. Arrow 1.

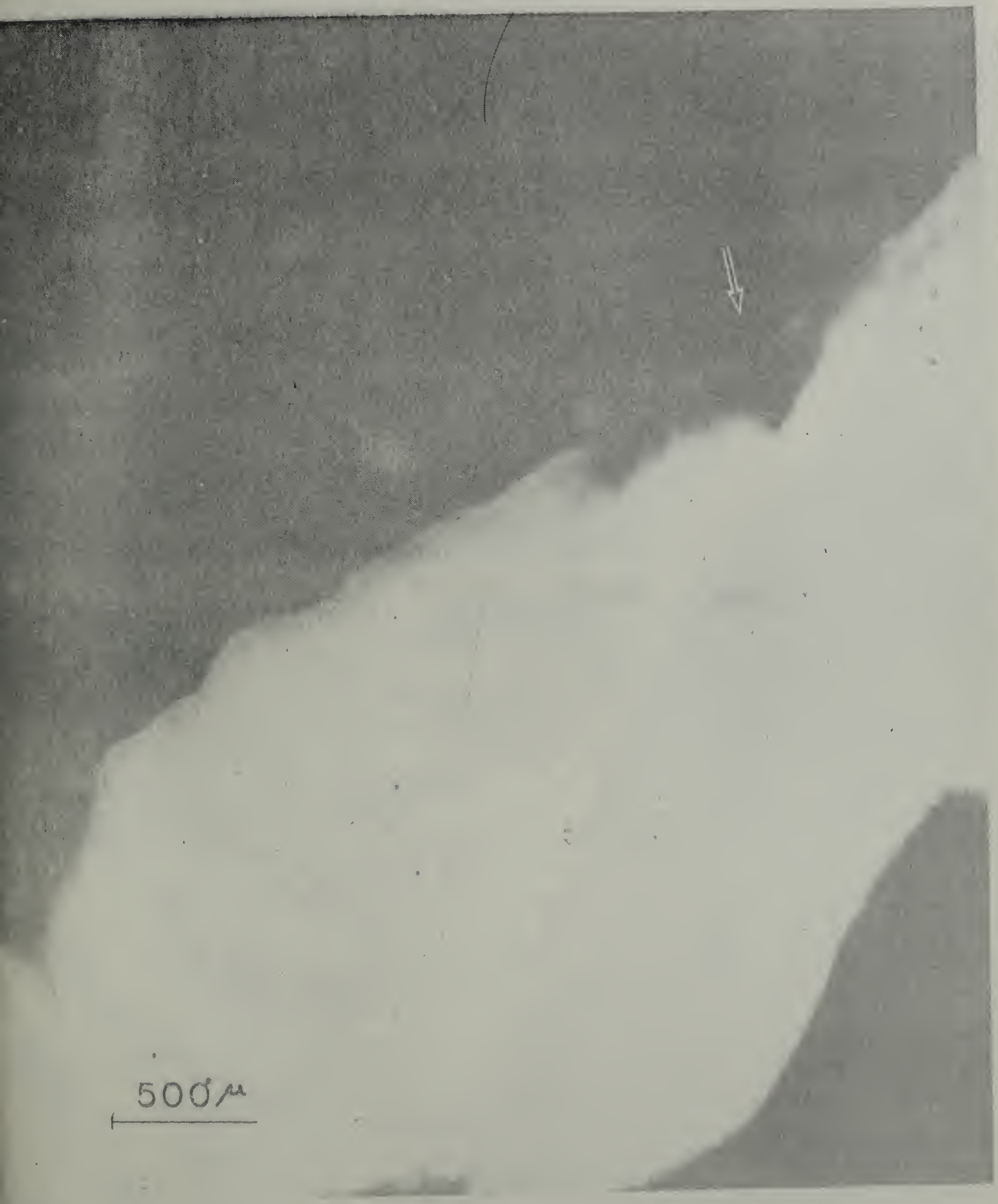

Figure 8. Microradiograph. Same area as Figure 7. Radiolucent areas indicate loss of mineral.

Figure 9. Polarized light, Nicols crossed. A shallow, localized lesion.

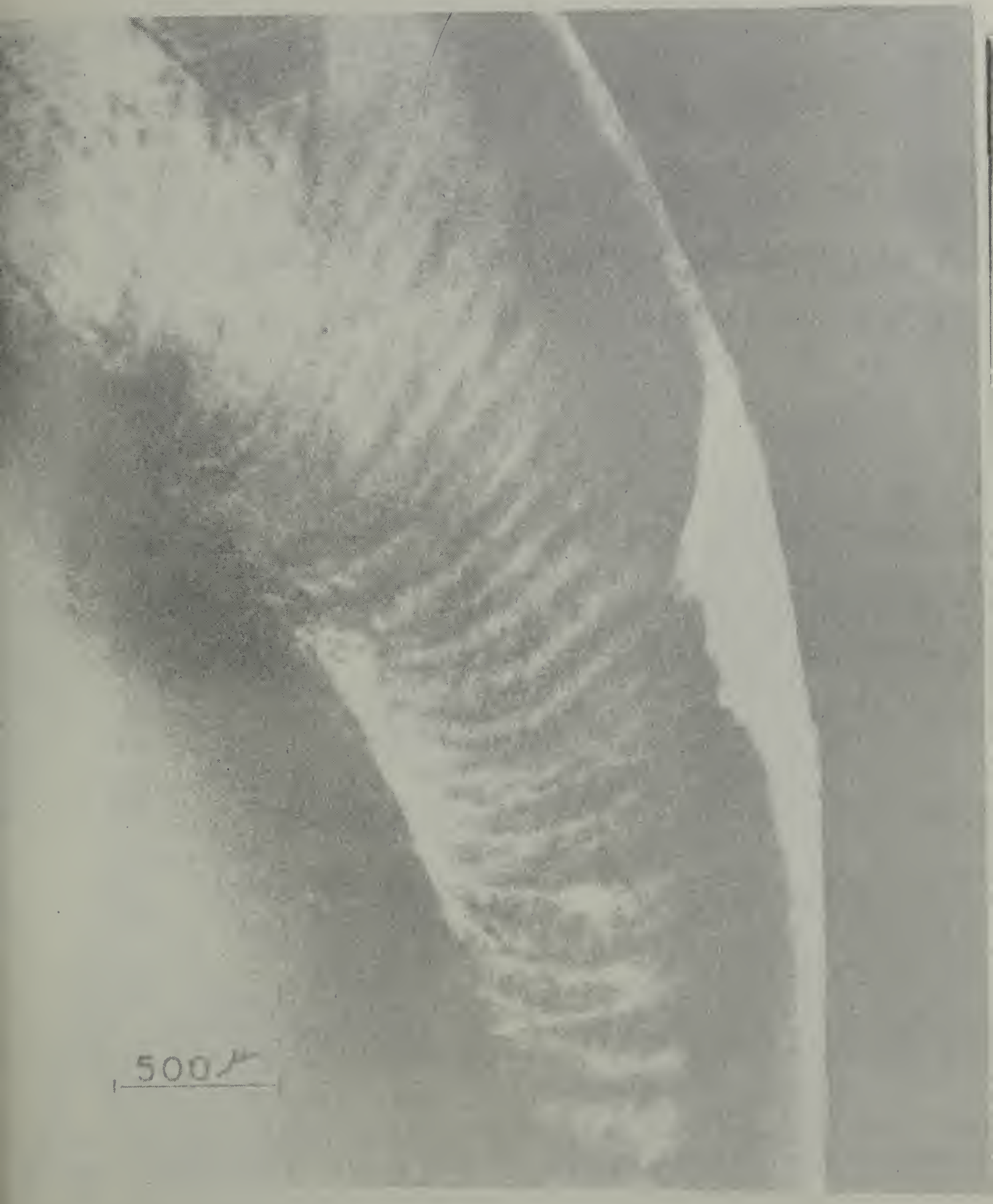
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Figure 11. Polarized light, Nicols crossed. The classic lesion ... a truncated cone.

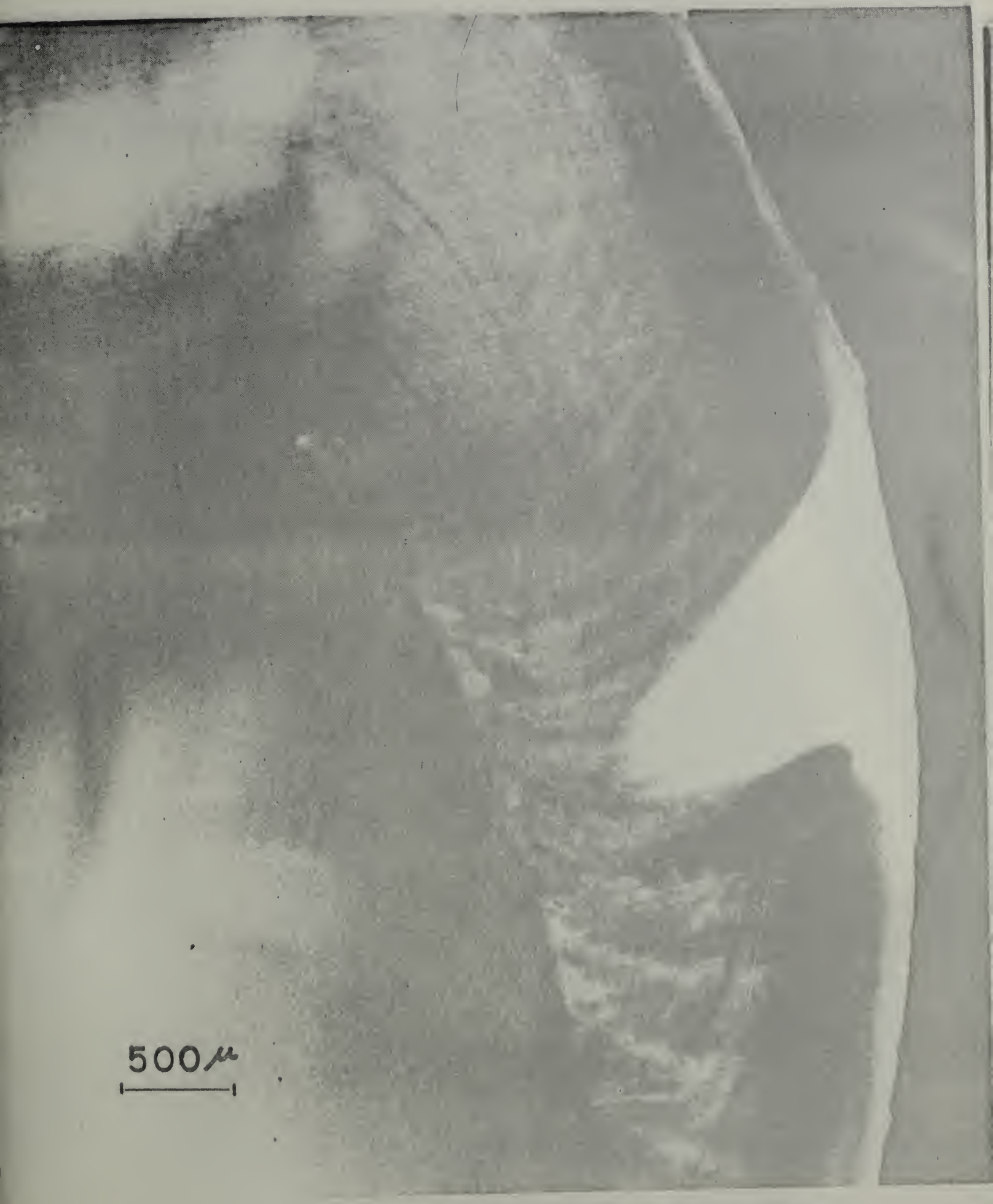


Figure 12. Reflected light (oblique). Same area as Figure 11.

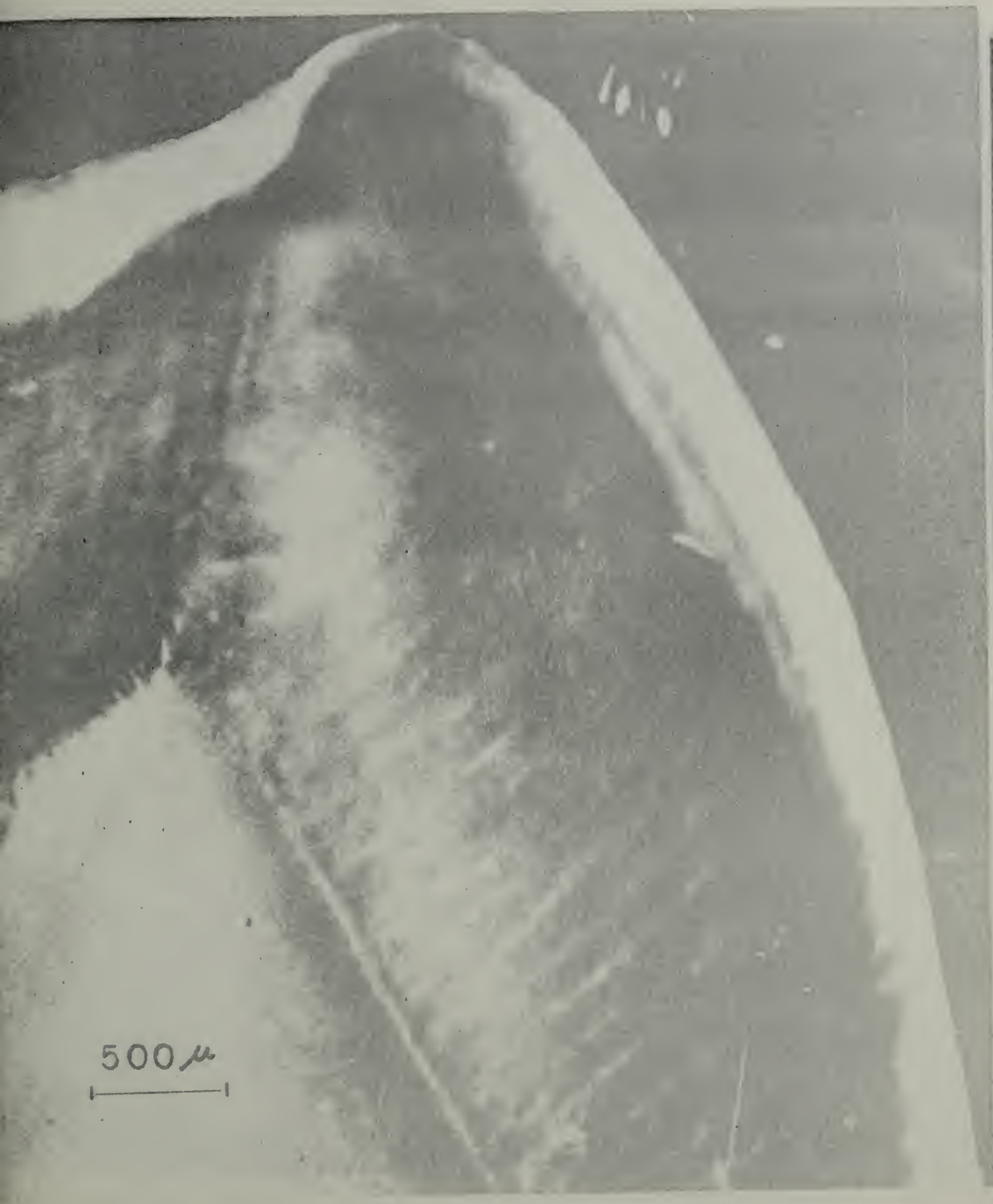
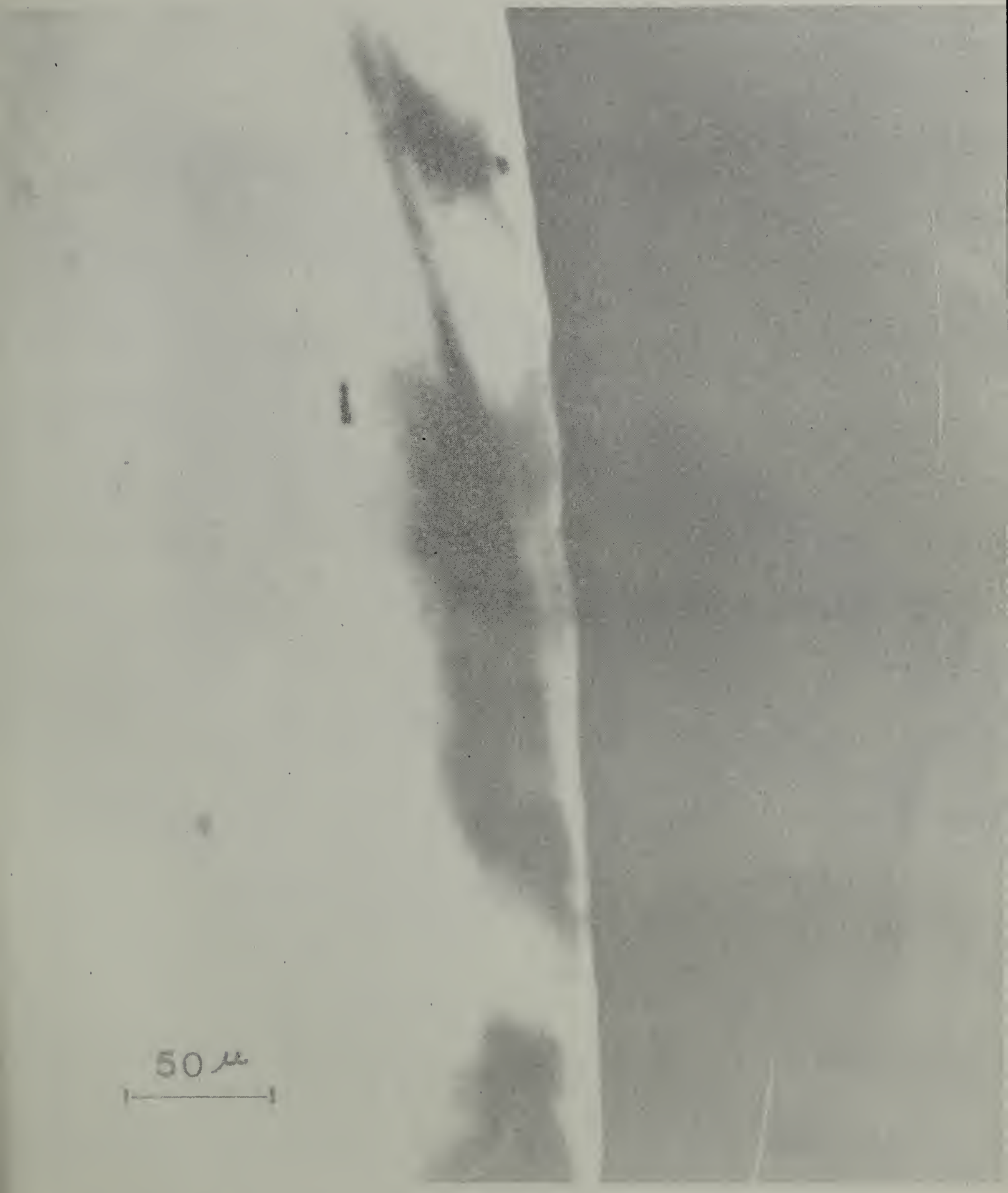


Figure 13. Reflected light (oblique) [illegible], [illegible] lesion on the buccal surface.

Figure 14. Microradiograph. The uniform progression of demineralization over a large area of a crown.

Figure 15. Microradiograph. A localized lesion with no preferred direction of attack.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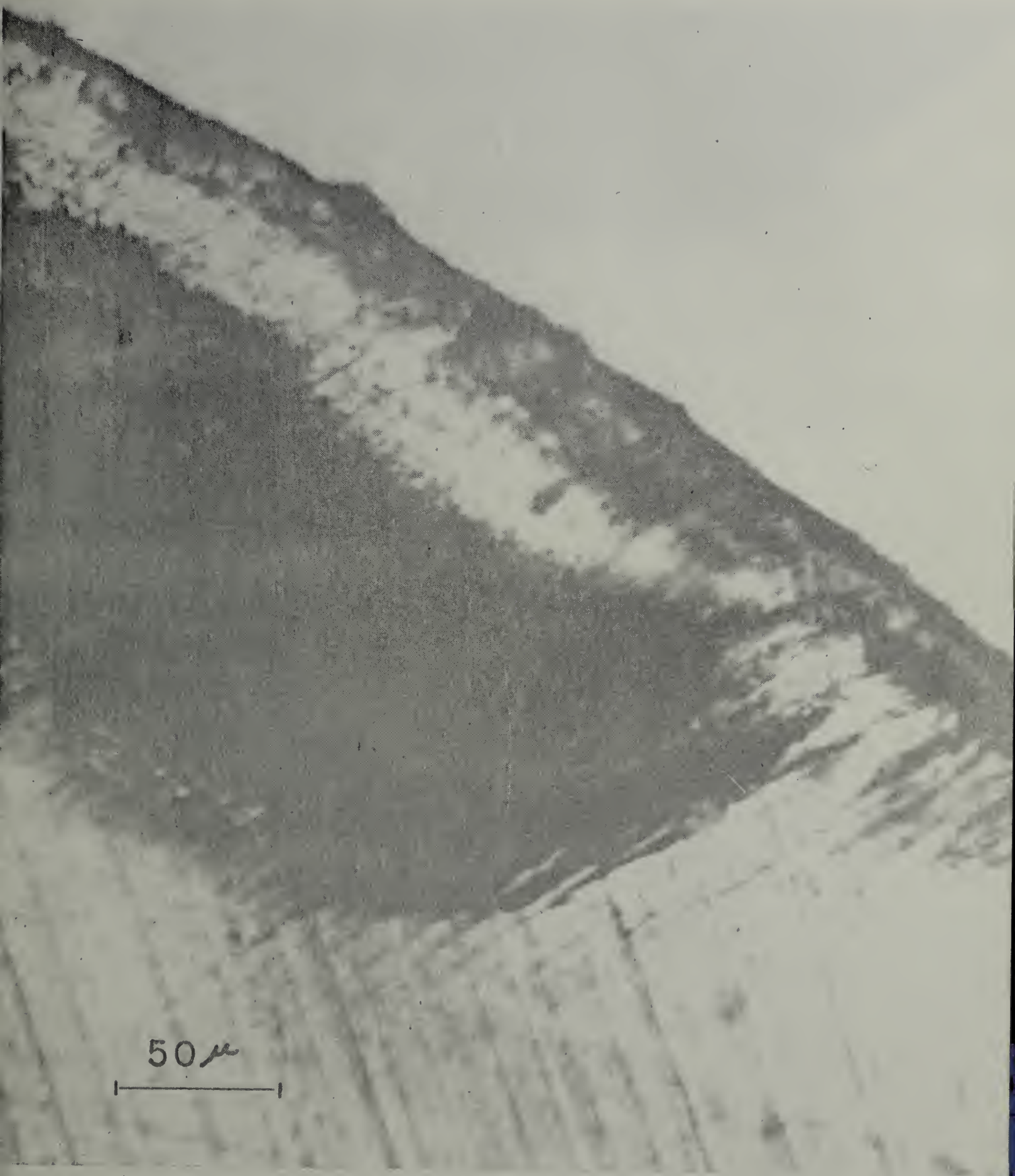

Figure 17. Polarized light, Nicols crossed, first order red. The surface layer above the lesion possesses the birefringence of a calcified area.

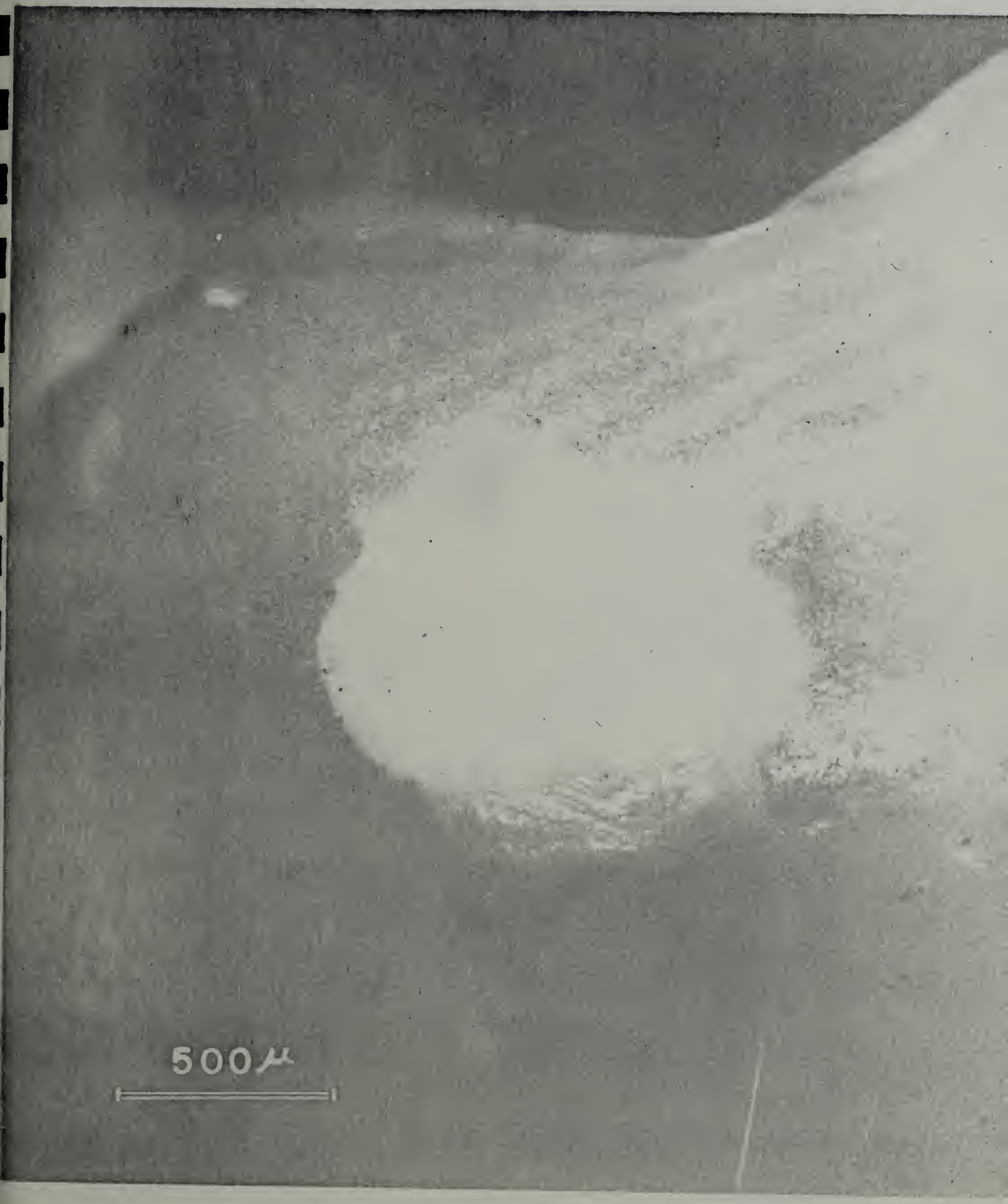

Figure 18. Reflected light (oblique). A distal contact point lesion.

Figure 19. Reflected light (oblique). Lesion in Figure 18 after being tapped with a chisel.

Figure 20. Reflected light (oblique). Thin section of the ethylenediamine tetraacetic acid decalcification.

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